EXHIBIT 1

Dkt. 62096/JPW/AJM/JCS

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants : David M. Stern, et al.

U.S. Serial No.: 09/687,528 Examiner: Shin-Lin Chen

Filed : October 13, 2000 Group Art Unit: 1632

For : A METHOD FOR INHIBITING NEW TISSUE GROWTH IN BLOOD VESSELS IN A PATIENT SUBJECTED TO BLOOD VESSEL INJURY

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Commissioner for Patents
P.O. Box 1450
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SIR:

DECLARATION UNDER 37 C.F.R. §1.132

I, Ann Marie Schmidt, M.D., hereby declare that:

1. I am a co-inventor named in the above-identified patent application.

2. I am a professor of surgical sciences at Columbia University in New York, New York. A copy of my curriculum vitae is attached hereto as **Exhibit A.**

3. I have reviewed and am familiar with pending claims 3-5 and 11-14 of the subject application. I understand that pending claims 3-5 and 11-14 provide a method for preventing exaggerated restenosis in a diabetic subject at risk of developing exaggerated restenosis which comprises administering to the subject a therapeutically effective

amount of soluble receptor for advanced glycation endproducts (sRAGE) so as to prevent exaggerated restenosis in the subject.

4. I also understand that pages 28-34 of the subject application set forth results from experiments involving fatty Zucker rats (also known as obese Zucker rats). In these experiments, it was shown that the blockade of RAGE in fatty Zucker rats by the administration of sRAGE suppresses exaggerated neointimal expansion (specification at pages 33-34). This finding provides a means for preventing excessive restenosis in subjects with diabetes (specification at page 34).

5. I have read and am familiar with the October 28, 2004 Office Action issued by the United States Patent and Trademark Office in connection with the subject application. I have also read and am familiar with the references of Muller, et al. ("Experimental Models of Coronary Artery Restenosis," J. Amer. Coll. Cardiol. 19(2):418-432 (1992)), Reilly, et al. ("Pharmacological and Mechanistic Aspects Concerning the Use of Heparin and β-Cyclodextrin Tetradecasulfate for the Treatment of Vascular Restenosis," Drug Dev. Res. 29(2):137-147 (1993)) and Lafont, et al. ("Why do animal models of post-angioplasty restenosis sometimes poorly predict the outcome of clinical trials?" Card. Res. 39(1):50-59 (1998)) cited by the Examiner in the October 28, 2004 Office Action.

6. I understand that in the Office Action, the Examiner rejected claims 3-5 and 11-14 as allegedly not enabled under 35 U.S.C. §112, first paragraph. Specifically, I understand the Examiner to assert that the specification does not provide enablement for a method for preventing exaggerated restenosis in a diabetic human subject by administering sRAGE to the subject.

7. I further understand that in support of this assertion, the Examiner cites Muller, et al., Reilly, et al. and Lafont, et al. as teaching that "successful application of restenosis treatments in small animal models is not predicative of success in other animals, particularly in humans" (Office Action at page 5).

8. I understand that each of Muller, et al., Reilly, et al. and Lafont, et al. discusses the use of rat models for the study of restenosis. I also understand that each of these references (i.e., Muller, et al. at page 428, Reilly, et al. at pages 143-144 and Lafont, et al. at page 52) indicates the disease-free arterial state of rat models as a disadvantage of such models in predicting human clinical outcome in treating restenosis. None of Muller, et al., Reilly, et al. and Lafont, et al. discusses the fatty Zucker rat model used in the subject application.

9. Fatty Zucker rats (i.e., those used for the experiments discussed in the subject application) are not healthy or normal rats. In support of this statement, I attach hereto as **Exhibit B** a copy of Park, et al. ("Neointimal

Hyperplasia After Arterial Injury is Increased in a Rat Model of Non-Insulin-Dependent Diabetes Mellitus," Circulation 104:815-819 (2001)). At page 815, Park, et al. state that the fatty Zucker rat is "characterized by excessive body weight, insulin resistance, hyperinsulinemia, and mild hyperglycemia" and "is a well-established model of type II diabetes."

10. Moreover, the arteries of fatty Zucker rats are diseased, as are those of diabetic humans. The response of fatty Zucker rat arteries following carotid balloon injury parallels the results observed in diabetic human subjects following carotid balloon angioplasty. As described by Park, et al., when subjected to identical degrees of balloon injury, compared to lean Zucker rats (nondiabetic), neointimal area was increased >2-fold in the diabetic obese Zucker rats. In parallel, medial cell proliferation was also higher after injury in diabetic obese Zucker rats relative to nondiabetic lean Zucker rats.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made herein on information and belief are believed to be true; and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the subject application or any patent issuing thereon.

Ann Marie Schmidt, M.D.

2/28/05

Date